

Letter: low population mortality from COVID-19 in countries south of latitude 35 degrees North supports vitamin D as a factor determining severity

EDITORS,

The recent editorial by Rhodes et al considered latitude and mentioned one mechanism that vitamin D is important in regulating and suppressing the inflammatory response of cytokines of respiratory epithelial cells and macrophages to various pathogens, including respiratory viruses and preventing cytokine storm and the subsequent acute respiratory distress syndrome (RDS).¹

It is appropriate to add the induction of the antimicrobial peptide cathelicidin with anti-viral action,^{2,3} and other beneficial mechanism of vitamin D including inhibition of the renin-angiotensin system (RAS) with inhibition of AT1R receptor, and stimulation of ACE2, the enzyme to which coronavirus binds and inhibits. This enzyme transforms angiotensin II into angiotensin (1-7), which is vasodilatory and hypotensive. This step, beneficial in these circumstances, is inhibited by SARS-CoV-2 and stimulated by vitamin D.

Lin reported that the renoprotective effect of calcitriol was due to the action on ACE, ACE2 and the ratio between both.⁴ Xu demonstrated in rats with RDS that calcitriol pre-treatment inhibited renin, ACE and angiotensin II, but induced ACE2, and resulted in clinical improvement.⁵ Gatera reviewed available evidence on vitamin D supplementation in animals and humans with RDS, and concluded that it was effective.⁶

In conclusion, both mechanisms may play a beneficial role in the action of vitamin D in COVID-19 infection—stimulation of the immune system and inhibition of RAS by stimulating ACE2.

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